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A REVIEW OF SUBMARINE ESCAPE TRIALS FROM 1945 TO 1970 WITH PARTICULAR EMPHASIS ON DECOMPRESSION SICKNESS

by

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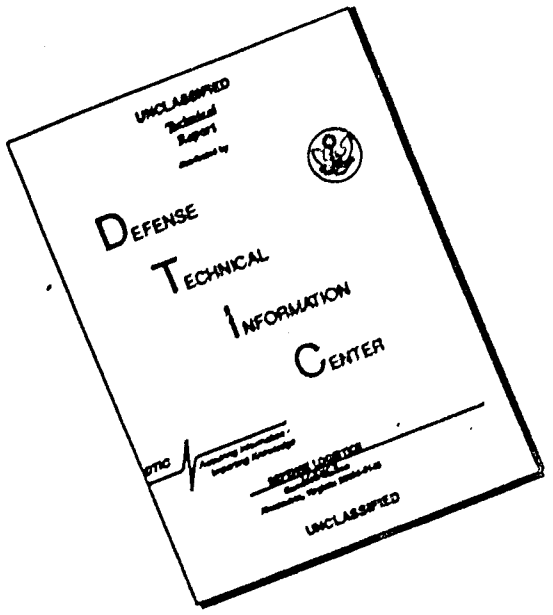

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SUMMARY

All simulated and actual submarine escape trials carried out by the Royal Navy from 1945 to the present time are described and tabulated. An attempt has been made to integrate these results.

A further attempt has been made to assess more precisely the risk of decompression sickness after submarine escapes. This risk has so far been determined entirely by trial and error. The theoretical behaviour of various tissues in the body during such escapes have been calculated and correlated with past results. A simple but effective formula is proposed to allow immediate assessment of the risk of decompression sickness after a particular escape.

The possible contribution of oxygen, and of the overloading of the 'fast' tissues with nitrogen, to the changing nature of decompression sickness after deep escapes is discussed.

The feasibility of even deeper submarine escapes and other future developments are briefly discussed.

CONTENTS

INTRODUCTION

Now that the "air breathing buoyant ascent" submarine escapes from 600 ft. have been successfully completed in open sea trials (Upshot V) it is necessary to consider whether deeper escapes are feasible and whether greater safety can be achieved in the present range.

The author felt that it would be useful to review all such simulated submarine escape trials and actual escape trials from submarines at sea carried out by the Royal Navy since the present series were initiated in 1945. The information has been gathered from various published and unpublished reports. A number of the earlier workers have been kind enough to seek out and provide further details from their original protocols. It is hoped that the bringing together of this widely scattered information into one document will help present and future workers to integrate and understand the results of numerous experiments over the last twenty-five years.

HANDLING OF DATA

Details of all goat experiments are given in Table I. Similarly, all simulated (chamber) or real submarine escapes carried out by Royal Naval personnel are given in Table II. There is a very wide range of depth, rate and type of compression, rate of decompression and time at maximal pressure. The number of subjects in each experiment also varied greatly. This number is, naturally, of great importance in interpreting the significance of any particular results.

The information is given in Tables I and II in the following manner. Column 1 gives the depth of the escape, column 2 the time at maximal pressure, column 3 the rate of compression and column 4 the rate of decompression. Columns 5, 6 and 7 are concerned with an attempt to provide an easy, albeit approximate, estimate of the amount of excess nitrogen contained in the subjects' tissues on surfacing after the escape procedure. This will be discussed at greater length later. Column 8 gives the number of subjects in each series and column 9 the number developing decompression sickness. Column 10 gives the individual details of each case of decompression sickness, column 11 the diagnosis and column 12 the final outcome.

As only two cases of decompression sickness occurred in the human exposures reported in Table II, columns 10, 11 and 12 are combined in the single column (9) where comments are made on certain aspects of the experiments.

Assessment of Risk. Returning to the figures given in columns 5, 6 and 7 in Tables I and II, it was felt that it might be useful to have an approximate estimate of the degree of risk to the subjects during these remarkably varied escape procedures. So far this has only been done by trial and error. The basis of the method used here is to merely calculate $\int_0^t$ Excess pressure. dt (termed SPdt from now on). This is the summation of the multiple of depth in feet (representing the increased pressure of nitrogen) x time in seconds during the whole of the exposure to increased pressure. This calculation will only give a measure of the nitrogen absorbed by the subject if the following conditions are satisfied :-

1. The body is taking up nitrogen throughout the exposure to increased pressure, including the whole period of decompression.

2. The rate of uptake of nitrogen remains proportional to the excess ambient pressure throughout the exposure.

These assumptions are patently not valid, particularly in the early series (see Table I) with slow compression and decompression and 2 to 7 minutes at maximal depth. Under these conditions the rate of nitrogen uptake for a given pressure will begin to fall considerably even while at high pressure and nitrogen will be excreted rather than absorbed during a considerable part of the decompression. In such cases SPdt will give a considerable overestimate of the extra nitrogen in the body and the risk of decompression sickness on surfacing.

As the series developed the escape procedure became a very rapid 'bounce' dive with rapid compression (20 seconds approx.) and a few seconds on bottom. The major exposure to increased pressure in such escapes is, in fact, at present, limited to about 8.5 ft. per second. during the ascent which is,

It is useful to consider a particular example of this type of escape as carried out from 500 ft. in the Upshot V trials (see Figure 1). In such a rapid escape (20 secs. compression of which 250 to 500 ft. only takes 5 seconds, 3 secs. maximum pressure, 60 secs. decompression) the circulation time becomes an important factor. If we assume that the blood takes 10 seconds to travel from the lung to the tissues, there will be a similar lag between the ambient pressure and the arterio-capillary blood gas tensions. Thus when the ascent commences the arterio-capillary blood gas tensions would be only 160 ft. sea water and when the arterio-capillary blood gas tension reaches 500 ft. the subject would have already ascended 60 ft. from the submarine. When the subject surfaces the arterio-capillary blood gas tension would still be 83 ft. of seawater.

Although it is tolerably certain that by far the greatest volumes of nitrogen will be absorbed during the decompression in such an escape, no previous attempts seem to have been made to estimate the nitrogen uptake of various tissues during the escape cycle. It is likely that in such a brief exposure to high pressure the 'very fast' (1-2 min. half saturation) and 'fast' (3-7 min. half saturation) are those mainly involved. The model selected for study was one kilogramme of 'fluid' (partition coefficient = 1) tissue with varying perfusion rates. It is generally agreed that the nitrogen uptake of 'fast' tissues is largely perfusion limited. Complete equilibration of nitrogen tension throughout the kilogramme of tissue and the effluent blood was also assumed. If the kilogramme of tissue is given a perfusion rate similar to that of the human brain (540 ml./Kg./min.) then the half saturation time at constant pressure is 80 seconds ($k = 0.52$). The uptake of nitrogen of such a tissue can be calculated during the various phases of the escape cycle and details and results of this calculation are given in Table III (Annex) and are illustrated in Figure 1. It is appreciated that, apart from the assumptions made, these calculations are not totally accurate but a number of very interesting points emerge.

In the 'very fast' model only 29% of the total nitrogen absorbed during the whole cycle is taken up during compression and at maximal pressure. Seventy-one per cent is absorbed during the decompression. Further, in this 500 ft. escape, nitrogen is absorbed until 4 seconds before surfacing.

In using SPdt as a measure of nitrogen absorbed and present in the body on surfacing it is assumed that the uptake is proportional to the ambient pressure throughout. One can easily calculate the nitrogen uptake of the tissue making no allowance for the increasing tissue nitrogen tensions during the escape procedure and these figures are also given in Table III. The calculated 'true' nitrogen uptake of the 'very fast' tissue is 76% of that as measured by SPdt.

This percentage will be much higher in less perfused or partially diffusion dependent tissues. For instance, if we take a 'fast' tissue with 150 ml./Kg./min. perfusion (half saturation time 5 minutes; k = 0.139), the calculated 'true' nitrogen absorption of this 'five-minute' tissue is 89% of that obtained when it is assumed that the nitrogen uptake is entirely proportional to the excess nitrogen pressure (SPdt technique). The reasons for this relatively small difference is the extreme brevity of the exposure and the very rapid fall of ambient pressure and nitrogen tensions just as the increasing tissue nitrogen tension is beginning to slow down absorption (see Figure 1). These calculations are also given in the Annex (Table IV). It will be noted that the 'five-minute' tissue is absorbing nitrogen throughout the escape from 500 ft. and that the final tissue nitrogen tension is only one atmosphere (Haldane ratio 2.29).

The possible rôle of 'very fast' and 'fast' tissues in decompression sickness after the escape procedure will be discussed later. There is no doubt that the use of a computer (in contrast to the author's slide-rule) to calculate the behaviour of different tissues during the escape cycle would greatly add to our understanding of the hazards of submarine escape. Meanwhile SPdt appears, on theoretical grounds, to be, for the time being, a useful approximate measure of the risk of decompression sickness after the escape. Figures 3 and 4 certainly bear witness to this.

Calculation of SPdt. Figure 2 shows a simulated escape (heavy line) from a depth D diagramatically. Time of compression (C), on bottom (T.O.B.) and of decompression (D.C.) are shown. If the rate of compression and decompression are constant, although they may be different, then it is obvious that (C/2 + T.O.B. + D.C./2) x max. depth equals SPdt. Columns 5 and 6 in Tables I and II show C/2, T.O.B., and D.C./2 separately and summated. SPdt is shown in column 7.

In all the early experiments a constant rate of compression was aimed at but not always achieved. In recent years in all the sea escapes there is not only a very rapid rate of compression but a doubling of pressure in constant time. This is not only favourable to ear clearing (early compression slower) but greatly reduces the time of exposure to the very high pressure and the resultant absorption of nitrogen. An accurate calculation of SPdt during compression can easily be made (C/3 x depth approximately) and should be used in future calculations.

Finally, all the results of the goat experiments are shown in Figures 3 and 4. In Figure 3 the 'equivalent time' at depth (C/2 + T.O.B. + D.C./2) is plotted against the maximal depth. The isopleths are the multiples of the ~~true~~ two values and allow SPdt to be noted in relation to each series. In Figure 4 SPdt is plotted against the escape depth. As SPdt is strongly influenced by the maximal depth there will be some inherent relationship. Nevertheless the figure demonstrates the value of SPdt, despite its definable shortcomings, as an easily calculable measure of safety.

In 78 out of the 90 series of exposures the number of goats employed was 5 to 30 Of the remaining 22 experiments, with four or less goats, 19, many of which were single exposures, were in the original 400-500 ft. studies (see Table I).

If even a single instance of Type II D.C.S. occurred in a series then it is represented in Figures 3 and 4 by a Type II D.C.S. symbol (●). The same holds with the series represented by the symbol for bends cured by recompression (▲) or bends recovering without recompression (Δ). If no case of decompression sickness occurred in the series then an open circle is used (o). Thus the degree of safety or danger is expressed in an all or none manner as, ideally, completely safe exposures are being sought.

Figure 5 illustrates in the same way the human simulated or actual submarine escapes which are detailed in Table II.

REVIEW OF ESCAPE SERIES

Donald, Davidson and Shelford, 1945 (1,2)

This work was carried out in the Admiralty Experimental Diving Unit between VE and VJ Days. The feasibility of this escape procedure, breathing air, rested on the safe immediate surfacing of goats after exposures of 15 minutes to air pressures of up to 150 ft. sea water. Reported instances of individual emergency free ascent escapes from over 200 ft. during the Second World War also gave rise to optimism (see Report of the Ruck-Keene Committee for details). In earlier work Kagiyama, 1934 (3) had shown that air divers could surface immediately after 15 minutes at 164 ft. without decompression sickness. Shilling and Hawkins, 1936 (4) had also shown that divers breathing air could surface safely after 37 minutes at 100 ft., 18 minutes at 150 ft. and 14 minutes at 185 ft.

Goats were used in the present study. They had long been employed in the study of decompression sickness, and work in many fields in the Admiralty Experimental Diving Unit during the war (mixture diving, surface decompression, etc.) had confirmed that the comparability with man, in this regard, was remarkable.

At this time the feasible rate of compression was considered to be 2 ft. per second. The time necessary at maximal depth was thought to be 2 to 3 minutes (exchange of signals, manipulation of hatch, possible ejection mechanisms). Observations showed that the rate of ascent without added bouyancy was also 2 ft. per second.

It will be seen (Table I) that successful runs occurred with simulated escapes from 150 ft. (3, 5 and 7 minutes on bottom), 200 ft. (3 and 5 minutes on bottom), and 250 ft. (3 and 5 minutes on bottom).

Incurable decompression sickness occurred after 7 minutes at 250 ft. (one out of four goats), 3 minutes at 300 ft. (three out of five) and 5 minutes at 300 ft. (one out of five).

A further series of simulated escapes from 300 ft. were carried out with the same rate of compression and decompression but using a 34% oxygen/66% nitrogen mixture. Two escapes with 5 minutes at 300 ft. and three escapes with 7 minutes at 300 ft. were completed with no signs of decompression sickness (2).

The assumption that the rôle of oxygen in decompression sickness could be ignored in superoxygenated mixture diving or submarine escape, or even in submarine escape breathing air from great depths, was also questioned at this time. Experiments were carried out (5, 6) which showed that very severe but transient decompression sickness could be caused by superoxygenated air (60% oxygen).

The tolerable rate of compression and possible dangers of nitrogen narcosis were also considered. It was shown that men could be compressed without discomfort at 6 ft. per second and that they were able to carry out cancellation tests during such a compression and at 300 ft. with reasonable efficiency (2).

Pratt and Taylor, 1946 (7)

These trials were carried out at the Royal Naval Physiological Laboratory after discussions between the Underwater Physiology Subcommittee and Admiral Ruck-Keene. Although they are reported as from 300 ft., they were actually from 330 ft. The 'danger zone' in the previous series was avoided and no important casualties occurred. The rate of compression and decompression was 2 ft. per second as in the previous series. Maximal time on bottom was 2 minutes. Three out of fourteen goats developed bends (Type I) in this exposure but only one required recompression.

Simulated escapes from this depth were also carried out (1 and 1.5 minutes on bottom) with a decompression rate of 4 ft. per second. No important difference was noted (see Table I).

Taylor, 1947 (8)

Taylor was asked by the Underwater Physiology Subcommittee to carry out longer exposures (time at maximal pressure) at 300 ft. The rate of ascent was again 4 ft. per second. His findings (see Table I) were very similar to those of Donald et al (1, 2).

If we examine the results so far obtained as plotted in Figures 2 and 3, the incidence of decompression sickness in 300 to 330 ft. range is reasonably consistent. Serious decompression sickness occurred when SPdt was over 990 hundred ft. sec. (h.f.s.). Bends requiring recompression occurred with 940 and 876 h.f.s. and bends not requiring recompression occurred with 841 and 706 h.f.s.

Taylor, 1947-8 (9,10)

During the late forties Taylor (9) showed that goats could safely carry out the simulated escape procedure from 220 and 300 ft. (4 ft. per second decompression) while submerged underwater. He further showed that unconscious anaesthetized goats could make similar simulated ascents while underwater without any untoward effects (10).

Wright (12) and Cowan (11), 1948-9

Human experiments were carried out at the Royal Naval Physiological Laboratory in relation to the respiratory behaviour during the ascent. Calculations suggested that there would be no important carbon dioxide accumulation in a 4 ft. per second ascent from 300 ft. Yet most subjects 'felt a great need' to inspire during 4 ft. per second decompression from 150, 200 and 250 ft. Only Wright continued to exhale throughout and he blithely completed 300 ft. escapes above and below water with 2 and 4 ft. per second rates of ascent. It is not generally realized that these were the first human simulated escapes from such depths (see Table II and Figure 5) and that Wright carried out the great majority of them. In the 300 and 330 ft. escapes SPdt figures of 399 to 549 h.f.s. were encountered but these are well within the safety zone at these depths as judged by goat experiments. No decompression sickness was encountered.

There was now considerable doubt as to whether most subjects could ascend from 300 ft. without inspiring and drowning, even if the rate of ascent were increased considerably.

Another most important contribution following the Ruck-Keene Committee was the introduction of the Submarine Escape Immersion Suit with a buoyancy stole. This, with 10 lbs. buoyancy, gave a rate of ascent of 4-5 ft. per second (13).

It had also been agreed that the 100 ft. tank should be constructed in HMS DOLPHIN for free ascent training.

In 1959 the HMS TRUCULENT tragedy high-lighted many of the great dangers of compartmental escape even from relatively shallow depths. The multiple hazards of compartmental escapes (decompression sickness, carbon dioxide poisoning, oxygen poisoning, compressed toxic fumes and gases) from greater depths were already well appreciated (2, 7). The need for free ascent training and definite positive buoyancy during the ascent was now even more clearly recognized.

Yet there was a remarkable pause during the next decade although buoyant ascent training began in 1953. Methods of absorbing carbon dioxide from the escape department during flooding (spray-flooding, refrigeration) were explored but without success. The use of oxy-nitrogen mixtures both in the built-in-breathing-system (BIBS) and in the submarine escape breathing apparatus was investigated and a 40% oxygen, 60% nitrogen mixture was recommended by the Subcommittee and adopted. However, the use of these mixtures was discontinued after a few years owing to difficulties in supply and proper maintenance out-weighing possible gains in safe escape depth.

The United States Naval Research Laboratory carried out work on increasing speeds of compression of human subjects and performed simulated escapes on air down to 450 ft. (no details available). Successful escapes (14) were accomplished by two subjects in open water from 302 ft. (keel depth 322 ft.). Compression time was 25 seconds, time at maximal depth 7 seconds and rate of ascent 5.7 ft. per second (53 seconds ascent). Assuming linear compression SPdt was 139 h.f.s., again, a very safe exposure at this depth.

It was not until 1962 that the Underwater Physiology Subcommittee strongly recommended that escapes should be carried out by the Royal Navy from submarines in open water. Rapid compression, which was now feasible, was also recommended even at the possible risk of rupture of the ear drums. At the same meeting another important development occurred. Trials of the Siebe Gorman hood were reported but with only moderate enthusiasm (15).

Upshot I Trials (HMS TIPTOE), 1962 (16)

These trials were carried out successfully in open water in August and September (see Table II). They were preceded by preliminary trials in HMS TIRELESS where the subjects were decompressed in the escape tower. High pressure air was used for compression, a geometric rate of compression was aimed at.

The deepest escapes from HMS TIPTOE were from 240 ft. (keel depth 260 ft.). Compression time was 30 seconds (pressure doubled in constant time), time at maximal pressure 27 to 49 seconds, and rate of ascent 5.5 ft. per second, with stole only, and 6.5 ft. per second with stole and hood. The maximal individual exposure was 30 seconds compression, 49 seconds on bottom and 58 seconds ascent. SPdt was 199 h.f.s., again, a very safe exposure. The subjects without a hood felt no desire to breath in during the ascent and no great preference was expressed for hood ascents (16).

In April 1963, the Submarine Escape Working Party of the Underwater Physiology Subcommittee was set up. Its remit was to make recommendations concerning open sea escape trials from 450 ft. and then, perhaps, from 600 ft. It was emphasized that the escape tower needed stream-lining. Even higher rates of compression, with doubling of pressure in constant time, were desirable and feasible. It was also considered necessary to improve the hood and its air supply during this very rapid compression. There was general agreement that time at maximal pressure should be only a few seconds.

Permission had been given by Flag Officer, Submarines for human trials to determine feasible rates of compression and the speed of onset and effects of nitrogen narcosis. These trials were to be followed by simulated escapes as recommended by the Subcommittee. The Royal Naval Physiological Laboratory had already achieved compression to 360 ft. in 18 seconds without untoward symptoms.

A series of simulated escapes using goats were also commenced. The results obtained were reported in UPS 232 (17, interim report) and in UPS 241 (18). The results given in Table I are those from the full report (18) by Barnard and Eaton. This work was complete before the Upshot IV Trials (July, 1965).

A useful and successful trial was carried out in Loch Fyne (HMS ORPHEUS) in October, 1964 from a keel depth of 200 ft. using the single escape tower, the new hood and inflation system and Mark VI Submarine Escape Immersion Suit. The rate of ascent was found to be 8 ft. per second.

Barnard and Eaton, 1963-5 (18)

A series of simulated escapes were carried out with goats from 300 to 500 ft. Compression time was 30 seconds and the rate of ascent 4 to 5 ft. per second. Time on bottom varied from 1.5 to 2.5 minutes. Five and ten minute decompression stops at 10 ft. were used as well as direct ascents. It is not possible to judge the protection afforded by the 10 ft. decompression stop procedure as against direct surfacing in this series as only one definite instance of severe decompression sickness occurred in 40 escapes. Only the direct ascents from 400, 450 and 500 ft. are shown in Table I (Series I).

In their second series (same time of compression and rate of decompression) all goats surfaced immediately. The 300 and 400 ft. escapes were uneventful, the time on bottom being about 30 seconds. In the 500 ft. escapes single studies were mainly carried out with increasing time on bottom. Two incurable Type II cases of decompression sickness occurred (see Table I and Figures 2 and 3) after exposures at maximal depth of 71 and 118 seconds. SPdt in these two instances was 740 and 920 h.f.s. respectively.

The human exposures were from 300 to 500 ft. (see Table II). The rate of decompression was 5 to 6 ft. per second. Time on bottom up to 400 ft. was 40 seconds. Time of compression (linear in time) was 20 seconds. If it was more prolonged the time on bottom was reduced by that amount. Barnard and Eaton first used 10 ft. stops, particularly after escapes from the greater depths and reduced the stop from 5 minutes to 1 minute before carrying out a direct ascent experiment. Seventeen successful direct ascent escapes were carried out from 350 ft. and ten from 400 ft. (maximal SPdt 360 h.f.s.).

At 450 ft., 19 successful escapes were completed with 20 seconds compression and 30 seconds on bottom. SPdt was 382 h.f.s. In the 500 ft. escapes 5 exposures were carried out with 20 seconds on bottom and 3 with 30 seconds, approximately, on bottom. The latter exposure is probably the most dangerous escape carried out by human subjects, SPdt being 442 h.f.s. One of the three subjects developed Type II decompression sickness (malaise, parasthesiae right arm, slurred speech, ataxia) which was rapidly cured by therapeutic recompression with air to 165 ft.

Upshot IV (HMS ORPHEUS), July 1965 (19, 20)

This trial was carried out in the open sea in July 1965. Maximal keel depth was 500 ft. (escape depth 475 ft.). The escapes, which were without untoward events, are detailed in Table II. Compression (pressure doubled in constant time) was achieved in 25 seconds, time on bottom was 4 seconds, rate of ascent 3 ft. per second. Maximal SPdt was 207 h.f.s., an extremely safe figure at this depth as judged by previous human and goat experiments (see Figures 2, 3 and 4). Upshot IV emphasized the remarkable success and potential of this method of escape. No important narcosis was felt by the subjects, thus confirming the findings of Bennett, Dossett and Ray (21). The hood inflation system through the buoyancy stole worked extremely well. Lieutenant Commander Todd reported that he considered that no special ability or lengthy training was required by the escapers.

After Upshot IV there was considerable debate in the Subcommittee and elsewhere as to whether experiments at greater depths were needed or justifiable. Nevertheless the Subcommittee decided to recommend that relevant research should continue so that animal data would be ready if a 600 ft. open sea escape was later proposed by the Executive.

Eaton, 1967 (i) (22)

The next series reported by Eaton from the Royal Naval Physiological Laboratory was a very important one. Escapes from 500 to 700 ft. were carried out, the time of compression (linear) being 30 seconds throughout. Two widely different rates of decompression were employed, 6 ft. per second and 15 to 20 ft. per second.

In the 500 ft. series, escapes with 30 and 60 seconds on bottom caused no decompression sickness at either rate of ascent. With 90 seconds at maximal depth there was a suggestive difference in favour of the more rapid ascent (see Table I). Unfortunately, both at 500 and 600 ft., as casualties began to appear with longer time on bottom, only the more rapid ascent was used.

However, there is one striking pair of series at 700 ft. With 45 seconds on bottom and a rate of ascent of 6 ft. per second, three incurable cases of Type II decompression sickness occurred in 6 escapes. With the same rate of compression and time on bottom and 15 ft. per second rate of ascent, there were no casualties in 8 escapes. The behaviour of 'very fast' (80 seconds) and 'fast' (5 minute) tissues have been calculated during these two escapes and are illustrated in Figure 6. It will be seen that the very rapid tissues achieved the same partial pressure of nitrogen (238 ft. s.w.) on surfacing in the slow and fast ascent escapes. The fact that there were no casualties in the fast ascent series suggest that 'very fast' tissues can tolerate a Haldane ratio of over 10 without causing manifest decompression sickness. The difference in the surfacing 'fast' tissue nitrogen tension with the two rates of ascent is not dramatic (123 and 94 ft. s.w.) but may well be critical. If one calculates the highest partial pressures in various tissues in Albano's study of maximal time at depth with immediate surfacing (23), making

due allowance for changes during the 20 metre/min. ascent, then the following figures are obtained : -

'Very fast' tissues (80 secs)	: 108 ft. nitrogen	: Haldane ratio 5.15
'Fast' tissues (5 min)	: 105 ft. nitrogen	: Haldane ratio 5.02
'Ten-minute' tissue	: 86 ft. nitrogen	: Haldane ratio 4.28

Albano's findings are very similar to those of Hawkins et al (24, men) and Eaton et al (25, goats). In these escapes the 'ten-minute' tissue nitrogen tension is far below these levels, but the 'five-minute' tissue nitrogen tension is above it in the escape with casualties (123 ft., ratio 5.7). It should also be pointed out that the distribution of nitrogen is very different after a plain dive at a constant depth and after a submarine escape, especially with a very short time on bottom. In the latter the 'very fast' (1-2 minutes) and 'fast' (4-6 minutes) tissue nitrogen tensions are far higher proportionately than in the slower tissues. Indeed, with this 'bounce' procedure this is not surprising.

Eaton, 1967(ii)(26)

Eaton completed another very large series of goat experiments later in the same year. This excellent study, which is reported in commendable detail, is again critically important. The time of compression was uniformly 30 seconds, time on bottom was 10 or 15 seconds and the rate of ascent was 6 ft. or 8 ft. per second. Escapes were carried out from 550 ft. working up to 800 ft. (see Table I). Eaton commenced with 30 goats in each simulated escape but casualties halved the number of the groups tested at the greatest pressures.

The pressure exposure is far more severe than would obtain in a submarine escape as the compression was linear and the time at maximal pressure was three to five times that used in Upshot IV. The results are given in detail in Table I and are illustrated in a separate section of Figure 3 to aid clarity.

A number of interesting findings emerged.

1. In all previous simulated escapes, using goats, Type II decompression sickness had been incurable or fatal despite careful therapeutic recompression with air. In this particular series there were 15 definite cases with paralysis, of which 12 recovered, 11 with recompression to 60 ft. on oxygen and 1 without recompression. Eaton remarks 'On recompression with oxygen the results are dramatic and the goat has usually recovered by 60 ft.'. In two instances (Cases 1 and 5) the signs were so transient that the diagnosis of decompression sickness was not sustained.

   There could be two possible explanations of these findings. Firstly, that the new therapeutic procedure (Goodman and Workman, 1965 (27))is far more efficacious than the previous procedure. In view of the very poor results with classical air therapeutic recompression in Eaton's previous 1967 series (see Table I) at not dissimilar depths there can be little doubt that this procedure is a considerable advance at least in this type of decompression sickness. The size of the bubbles is reduced to one-third and there is no added nitrogen supersaturation in the presence of bubbles in the body. As this treatment is now used, providing there is rapid relief, to treat decompression sickness in human subjects after escapes it would be of great interest to determine whether the oxygen is really critically important and whether the method is still effective when 'slower' tissues are involved.

2. Even allowing the improvement of the therapeutic procedure, the very rapid recovery of most of the cases immediately after or during recompression to only 60 ft. and the spontaneous recoveries without recompression suggest that, paradoxically, the disease is less severe at these depths and that we are encountering a new type of decompression sickness.

The author suggested many years ago (6) that mixed oxygen-nitrogen bends may occur in this type of submarine escape from great depths while breathing oxy-nitrogen mixtures or even air. This is an attractive hypothesis in relation to some of these findings as it would explain the transient nature of the Type II decompression sickness in some instances and the rapid response to mild recompression in others. This has been demonstrated in decompression sickness caused by super-oxygenated air (Donald, 1945; Hempleman (28), 1969).

Yet if we again apply the tissue model to an actual 800 ft. simulated escape in this series (30 seconds compression, 15 seconds on bottom 6 ft. per second ascent) and assume that the oxygen uptake, like the rate of perfusion, is similar to that of the human brain (33 ml./Kg./min.) we find that the excess of dissolved oxygen perfusing into the tissue over the oxygen used is very small. The calculated net figures of excess dissolved oxygen (ml. per kilogramme) are given in Figure 7 against the blood gas tension plot. It will be seen that the highest figure is 18.1 ml./Kg. of tissue (0.77 atm.) and that the oxygen uptake exceeds the intake at 420 ft. As the goat ascends further the amount of excess oxygen becomes trivial and finally 'negative'.

The same calculations were carried out for a 1000 ft. escape with 15 seconds on bottom (30 seconds compression, 15 seconds on bottom, ascent 8 ft. per second) and the highest excess tissue oxygen tension was only 1.0 atm. at 560 ft. After this, the uptake exceeded the intake and the excess oxygen fell to zero in the last few feet of the ascent.

It can be said that the tissue studied has a very high oxygen uptake but, in general, the rate of blood perfusion of a tissue is proportional to the degree of metabolic activity. Nevertheless it is possible that some tissues have higher perfusion/metabolism ratios and may have significantly raised oxygen tensions on surfacing in these conditions. For instance, the kidney has a rate of blood flow which is disproportionate to its oxygen demands and this may be a source of danger. More research is needed before a more definite opinion concerning the rôle of oxygen can be given.

There is another possible explanation of the changing characteristics of the decompression sickness as time on bottom becomes briefer and the escapes deeper. We may be dealing with a decompression sickness that only involves the 'fast' (4-6 min.) tissues. When discussing the first escape series, the author attributed their success to the old adage 'Easy come, easy go' (Donald, 1948 (2)), but there must finally be a limit to the 'go', particularly if there is a phase discontinuity of nitrogen.

Figure 7 illustrates one of the escapes from Eaton's 1967 (ii) series from 800 ft. where decompression sickness (bends) occurred in only 1 out of 13 goats. The nitrogen tension in the 'very fast' tissues on surfacing was 230 ft. (Haldane ratio 9.9). Evidence has already been given that these very high nitrogen tensions on surfacing are tolerable in the 'very fast' (1-2 min.) tissues. Again we find the 'five minute' tissue nitrogen tension on surfacing

just at the critical level (104 ft. Haldane ratio 5.00). The nitrogen tension in the 'ten minute' tissue on surfacing is 56 ft. (Haldane ratio 3.14) and this is generally considered to be below the tolerable level (4.25).

In Eaton's 1967 (ii) study there is a 'paired escape' from 800 ft. with similar times of compression and on bottom but with a 6 ft. per second ascent as opposed to the 8 ft. ascent in the escape just discussed (see Table I). The casualties in this escape were far more severe. If the behaviour of the tissues is calculated during this escape the 'very fast' tissue tension on surfacing is the same as in the 6 ft. ascent escape, but the 'five minute' tissue nitrogen tension on surfacing is significantly higher (114 ft., Haldane ratio 5.35). This again suggests that the 'five minute' tissues are the most critical in this type of escape. The transient or easily curable nature of the decompression sickness may be partly due to the small amount of nitrogen involved (see Table IV).

3. Finally, if this series of escapes is studied in Figure 3, it will be seen that they 'move across' the time-pressure dosage isopleths (SPdt) of 500 and 750 h.f.s. It can also be seen that at these depths the critical SPdt is 500 h.f.s. The one exception to this in the 312 exposures with an SPdt below 500 h.f.s. was not classified as a bend by Eaton as recovery was so rapid.

Upshot V (HMS OSIRIS), July 1970 (29)

In June 1969, Flag Officer, Submarines approached the Royal Naval Personnel Research Committee on the feasibility of deeper escape trials in the open sea from depths up to 600 ft. The Underwater Physiology Subcommittee considered this to be entirely feasible but proposed that the following experiments should be done before embarking on such escapes.

1. No animal experiments had been carried out with minimal time on bottom in this range. It was therefore proposed that the range of safety of such escapes should be tested to the limit in the following exposures:-

From 650, 700, 750, 800, 850, 900 and 950 ft. using twelve goats at each depth with 25 seconds compression, 3 to 5 seconds at maximal pressure and an ascent rate of 8.5 ft. per second. Compression was linear with respect to time as the pressure profile of sea escapes could not yet be reproduced in chambers. These were duly carried out and no untoward signs occurred up to 800 ft. After the 850 ft. exposures all goats 'looked very unwell' but developed no specific signs and survived unharmed. After the 900 ft. exposure all animals appeared unwell and two developed Type II decompression sickness which remitted without recompression. In the final 950 ft. escape ten goats appeared unwell but recovered and two developed severe Type II decompression sickness which necessitated their dispatch (see Table I).

2. After these entirely successful escapes up to 800 ft. the Underwater Physiology Subcommittee recommended human chamber escape trials as follows:-

> Escapes from 500, 550, 600 and 625 ft. with 20 seconds compression, 3 seconds at maximal pressure and a rate of ascent of 8.5 ft. per second.

These were successfully accomplished without any untoward events (see Table II and Figure 4). The maximal risk escapes from 625 ft. had a calculated SPdt of 312 h.f.s.

The sea trials were carried out successfully in July 1970, the deepest escapes being from 586 ft. (keel depth 600 ft.). One important incident occurred. The trials' officer suffered 'impairment of vision and balance' after an ascent from 505 ft. He made a rapid and complete recovery with recompression to 60 ft. on oxygen. SPdt was 237 h. f. s. and judging by other human and goat exposures this would have been expected to be an extremely safe escape. A detailed medical report is still awaited.

Decompression Sickness in Human Subjects after Escapes

It is worth briefly considering the present position with regard to post-escape decompression sickness in human subjects.

Although goats differ from men in their handling of nitrogen after prolonged exposures there has been a remarkable similarity between men and goats after relatively short exposures (see Barnard and Eaton, 1965 (18)) and, until recently, it was reasonably assumed that the same applied to these escape procedures. Nevertheless a considerable margin of safety was allowed when moving from goat to human exposures.

Only two subjects have suffered decompression sickness after chamber or sea escapes. The first has already been mentioned. This subject, who was the oldest member of the group and supervising officer, suffered a Type II bend after a chamber escape from 500 ft. The exposure was fairly severe (SPdt 442 h. f. s.). This officer had carried out four chamber escapes from 450 or 500 ft. (SPdt 380-400 h. f. s.) over a period of four weeks prior to the episode. After the event he carried out an escape from 450 ft. (SPdt 340 h. f. s.) one week later, an escape from 450 ft. (SPdt 280 h. f. s.) three months later and another escape from 450 ft. (SPdt 230 h. f. s.) three weeks later. In this last escape he again developed Type II decompression sickness with 'heaviness' and parasthesiae affecting the left arm, buttock and thigh. His previous attack had been on the right side. Symptoms and signs (loss of knee bicipital reflexes) did not clear until he was compressed to 165 ft. on air. He made a complete recovery.

The recent incident in Upshot V has some important similarities. It occurred at a level of exposure where bends were not expected. The subject was, again, the supervising officer who had been exposed to high pressures a great deal. Further, he had carried out a considerable number of sea escapes in the previous few days. He was the oldest member of the group. He stated that he had suffered a not dissimilar attack (visual scotoma) while boxing some years before. He is a migrainous subject.

Thus both men had carried out a number of escapes before the event and both were the oldest subjects. As Todd aptly remarks (29) 'repetitive escaping is an unexplored subject'. As we would expect, officers have certainly insisted on doing more than the average stint in escape trials. It is highly probable that bubbles are liberated in all these deep escapes and, in our present state of knowledge, it might be wise to strictly limit the total number of deep escapes by one subject during the whole of his service career. The question of age may be also important as these officers, with due respect, had passed their physiological prime.

If there are to be future deep escape trials then these matters will certainly need resolution.

Finally, migraine in a subject, although it may well have been irrelevant in the Upshot V incident, is not without danger in extreme hyperbaric work. Stress precipitates migraine and if the subject is under pressure during the period of local cerebral vasodilatation then there would be a real risk of abnormal quantities of nitrogen,

and even oxygen, being absorbed by the part of the brain involved. It appears that we have stumbled over yet another contra-indication to high pressure work.

Eaton and Hempleman, 1971 (30)

Since the submission of the first draft of this memorandum to the Subcommittee, the above workers have issued a preliminary report on the use of decompression stops after simulated escapes by goats from very great depths (up to 1200 ft.).

Three minute 'stops' at 30 ft. allow safe escapes to be made from 900 and 950 ft. (30 seconds linear compression, 5 seconds on bottom, ascent at 8.5 ft. per second). With 4 minute 'stops' at 40 ft. escapes can be made from 1100 ft. without casualties. K.E. Tayler (DGS) has intimated that escape suits capable of withstanding positive pressures of up to 50 ft. may well be feasible.

These workers subscribe strongly to the author's proposition that oxygen may be playing an important rôle in the decompression sickness encountered after very deep escapes.

If one calculates the orders of nitrogen tension after a 1000 ft. escape they are as follows : -

'Very fast' tissues	(1.3 min.)	: 265 ft.	: Haldane ratio 11.1
'Fast' tissues	(5 min.)	: 124 ft.	: Haldane ratio 5.75
'Ten-minute' tissue		: 68 ft.	: Haldane ratio 3.6
'Twenty-minute' tissue		: 16 ft.	: Haldane ratio 1.6

This, as already emphasized, is an entirely different pattern to that encountered after exposures to relatively constant increased air pressure. It is unlikely that the '10 and 20 minute' tissues are involved in the decompression sickness in this situation as they are well below the accepted critical levels. The 'very fast' tissues, although they have these remarkable nitrogen tensions, do not appear to be critical as calculations show that they would drop dramatically to 104 ft. (ratio of 5.0) after 3 minutes at 30 ft. Yet with this 'stop' after a 1000 ft. escape 20% of goats developed bends.

The '5 minute' tissue nitrogen tension would drop to 85 ft. (ratio 4.25) after a 4 minute stop at 40 ft. and to 90 ft. (ratio 4.45) after a 3 minute stop at 30 ft. These figures are not conclusive but considering the many assumptions made in these calculations, the possible reinforcement by oxygen and the added effect of possible phase separation of gas they are highly suggestive that the critical tissues are in the '5 minute zone'.

CONCLUSION

The whole purpose of this work over the last twenty-six years has been to make submarine escape possible from considerable depths.

The achievement has been remarkable. Escapes have been successfully carried out from a submarine depth of 600 ft. We can state, with reasonable confidence, that safe escapes can be achieved from 750 ft. There would be a majority of survivors from 900 and 950 ft. but there would certainly be casualties who would require urgent recompression. The problem of escapes from anywhere on the continental shelf has therefore been solved. The demands on the escaper are not great, although no one would pretend that escaping from a submarine by any method, particularly after an accident or hostile action, is anything but unpleasant. Nevertheless all that the escaper has to do is to connect his

hood inflation system and see that it remains connected during compression. The rapidity of the procedure is such that from the time of commencing compression to surfacing in a 600 ft. escape is 95 seconds, the escaper having briefly passed through a pressure of over 19 atmospheres absolute and back to one atmosphere.

The two cases of decompression sickness in the human escape trials must not be forgotten. We must accept that they may occur sporadically in fit men after deep escapes that are generally considered to be safe. Age is almost certainly an important factor. It is possible that even these very occasional casualties will disappear if repeated deep escapes by the same individual are avoided in the future.

Recompression must, when possible, be always available. The method of recompression introduced by Eaton for post-escape decompression sickness is certainly an important advance.

With the modern escape pressure profile (rapid 'geometric' compression, minimal time on bottom, ascent at 8.5 ft. per second) SPdt is, perhaps, more by good luck than good judgement, an excellent method of assessing the degree of safety. It can be calculated for any escape, without even a paper and pencil in a few seconds and gives a good measure of the '4-6 minute' (and longer) tissue nitrogen tensions on surfacing.

Throughout this work it has been the deliberate policy of the Underwater Physiology Subcommittee to exploit the most simple method (air-breathing, immediate surfacing) to its absolute limit before considering any more complex alternatives. This limit has now been reached. Compression is now so rapid that ear drums have been ruptured on a number of occasions. The episodes are apparently painless and the drum heals quickly. Perhaps compression can be further shortened but the advantage will be very slight except at enormous depths, say over 1500 ft., where it would probably be essential.

Time on bottom is already minimal. The rate of ascent cannot be further increased without propulsion. This is not feasible at present and very rapid ascent may introduce new hazards. In the not unlikely event of small propulsion units becoming available they will certainly be of great use, particularly in very, very deep escapes (below 1500 ft.) as an escaper could accelerate during his early ascent and decelerate as he approaches the surface. Automatic drogues have already been suggested for this latter purpose (29). These possible developments, reminiscent of astronauts, emphasize the great simplicity of the present method.

Super-oxygenated air which allowed much longer exposures at 300 ft. appeared at one time to have great promise. However, with the modern type of escape, air is safe up to about 700 ft. and at greater depths oxygen rich mixtures would almost certainly cause mixed oxygen/nitrogen bends.

The use of decompression stops by developing a suit to retain up to 1.5 atmosphere internal pressure for several minutes will certainly increase the depths from which safe escapes can be made. Incidentally, the rate of fall of tissue nitrogen in the subject is greater at, say, a 30 ft. stop than at a 40 ft. stop and the use of the more shallow stops should be fully exploited.

Another problem which needs clarification is the rise of pressure in the submarine before and during the escapes. It is obviously most important to keep the initial nitrogen in the escaper's body to a minimum.

There is still the ever-present risk of aero-embolism whether a man is escaping from 30 or 1000 ft. It is fortunately very rare and occurs most frequently in training where large groups of men are first exposed to these conditions. Scrupulous observation and immediate recompression usually prevent serious consequences. More knowledge is needed of the minor abnormalities of the lung which occasion this highly dangerous series of events, particularly with a view to their detection during routine medical examinations.

Finally, although this review is almost exclusively concerned with decompression sickness after submarine escape, the author and his colleagues are well aware that without the ingenuity and skills of many experts in all aspects of naval science and the energy and enthusiasm of the executive officers responsible for the escape trials, the present method of submarine escape would have remained a laboratory dream.

ACKNOWLEDGEMENTS

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REFERENCES

1. DONALD, K W and DAVIDSON, W M. Admiralty Experimental Diving Unit Report. XVIII, 1946.

2. DONALD, K W, DAVIDSON, W M and SHELFORD, W O. J Hyg (Univ Camb) 46, 176, 1948.

3. KAGIYAMA, S.J. Kumamoto med Soc, 10, 562, 1934.

4. SHILLING, C W and HAWKINS, J A. US Nav Med Bull Wash, 34, 47, 1936.

5. DONALD, K W. M D Cambridge, 1945.

6. DONALD, K W. J Appl Physiol, 7, 639, 1955.

7. PRATT, C L G and TAYLOR H J. UPS 78, 1947.

8. TAYLOR, H J. Reported from UPS 137, 1953.

9. TAYLOR, H J. UPS 79, 1947.

10. TAYLOR, H J. UPS 90, 1948.

11. COWAN, S L, WILSON-DICKSON, W. and WRIGHT, H C. UPS 82, 1947.

12. WRIGHT, H C. UPS 113, 1950.

13. GOODENOUGH, F C and BARTLETT, P. UPS 100, 1948.

14. BOND, C F, WORKMAN, R D and MAZZONE, W F. US Nav Med Lab Research Report, 346, 1960.

15. HAMLYN, L D and MACKAY, D E. UPS 204, 1962.

16. HAMLYN, L D and PARSONS, M H. Upshot I, UPS 229, 1962.

17. BARNARD, E E P. UPS 232, 1964.

18. BARNARD, E E P and EATON, W J. UPS 241, 1965.

19. TODD, M R. Operation Upshot IV. Report to Flag Officer, Submarines, 1965.

20. HAMLYN, L D and TAYLER, K E. Upshot IV, Report from Director General, Ships, to UPS, UPS 263, 1967.

21. BENNETT, P B, DOSSETT, A N and RAY, P. UPS 239, 1964.

22. EATON, W J. UPS 264, 1967. (i).

23. ALBANO, G. Medicina dello sport, 1, 6, 1961.

24. HAWKINS, J A, SHILLING, C W and HANSON, R A. US Nav Med Bull Wash, 33, 3, 1935.

25. EATON, W J and HEMPLEMAN, H V. UPS 209, 1962.

26. EATON, W J. UPS 268, 1967 (ii).

27. GOODMAN, M W and WORKMAN, R D. US Nav Exp Diving Report, 5-65, 1965.

28. HEMPLEMAN, H V. UPS 283, 1969.

29. TODD, M R. Upshot V. Report to Flag Officer, Submarines, 1971.

30. EATON, W J and HEMPLEMAN, H V. Royal Naval Physiological Laboratory, Report 3/71, 1971.

Note 1. The 'UPS' prefix indicates papers or reports prepared for the Underwater Physiology Subcommittee of the MRC Royal Naval Personnel Research Committee.

Note 2. Reports quoted are not necessarily available to members of the public or to commerical organisations.

Annex to:-
UPS 290

Calculation of Tissue Nitrogen Tension during Escape Cycle and on Surfacing

Only two tables (III and IV) are given to illustrate the method used. These show the calculated uptake of nitrogen of an '80 second' tissue and of a '5 minute' tissue during an escape from 500 ft. (compression 20 seconds (geometric), time on bottom 3 seconds, ascent 8.3. ft. per second).

Note that in these tables the gas tensions in the arterio-capillary blood is being specified and therefore the times of the compression, bottom and decompression phases are all delayed 10 seconds to allow for the lung-tissue circulation time (see Figure 1). The time of surfacing is the true surfacing time as interest lies in the nitrogen tissue tension at this moment.

Throughout the paper the model studied is one kilogramme of 'fluid' tissue (partition coefficient unity). It is also assumed that the absorption of nitrogen is entirely perfusion limited and that there is complete equilibration of nitrogen tension throughout the kilogramme of tissue and the effluent blood. This is generally considered permissible with 'very fast', 'fast' and 'relatively fast' tissues (1.3 to 10 minutes half-saturation time).

The total excess of nitrogen carried by the incoming blood at tensions above the tissue tension is calculated for each specified period in ml. It is then partitioned between tissue and blood. The resultant tissue uptake of nitrogen, which is cumulative, is then converted to tissue nitrogen tension stated first in atmospheres (solubility of nitrogen employed is 12.2 ml./1./760 mm. Hg. nitrogen tension) and then in feet of sea water. Thus the tissue nitrogen tension can be used to repeat the exercise during the next period.

When the blood-tissue nitrogen gradient is reversed, the total excess nitrogen in the kilogramme of tissue is calculated and this is partitioned between the tissue and the blood. The partition factor to determine the loss of nitrogen to blood from the tissue is one minus the usual factor (see Table III). The model studied in Table III has a perfusion rate of 540 ml./Kg./min. which is similar to that of the human brain. Half saturation time at constant increased pressure is 80 seconds (k = 0.52). The calculated results are plotted in Figure 1. More detailed calculations show that the blood-tissue nitrogen gradient becomes zero four seconds before surfacing and that the nitrogen lost during this period is almost negligible (0.2 ml.).

In the lower half of Table III the tissue nitrogen uptake is calculated as if it remains entirely proportional to the tension of nitrogen in the incoming blood and the rise of nitrogen tension in the tissue is ignored. This assumption is made when SPdt is used as a measure of nitrogen uptake during the escape. The uptake of nitrogen allowing for the rise of tissue nitrogen tension is 76% of that when the simple SPdt procedure is adopted.

In Table IV a similar exercise is carried out with a model where the perfusion rate is 125 ml./1./min. At a constant increased pressure the time of half-saturation is 5 minutes (k = 0.139).

The notable findings are that nitrogen is taken up by the tissue throughout the escape procedure. The total uptake is just over one-third of the uptake by the '80 second' tissue. The difference between the nitrogen uptake, allowing for increasing tissue nitrogen tensions and the uptake when this factor is ignored (SPdt calculation) is

remarkably small. The SPdt figure is 93% of the former figure. As it appears probable (see main text)that the '5 minute' tissues are particularly important in the genesis of decompression sickness in air escapes this may account for the surprisingly good correlation between SPdt and the occurrence of decompression sickness, especially in the rapid 'bounce' escapes at greater depths (see Figures 2 and 3).

It should be emphasized that in reality the concept of '5 minute' or '80 second' tissues does not necessarily apply to a particular continuous tissue. The perfusion rates of any specific tissue or organ vary greatly in different parts of that organ and small areas of tissue with particular time constants may be scattered through many parts of the body.

This would account for the great variety of signs or symptoms even when 'fast' tissues are mainly involved, although the central, and perhaps peripheral, nervous system appear most vulnerable in this regard.

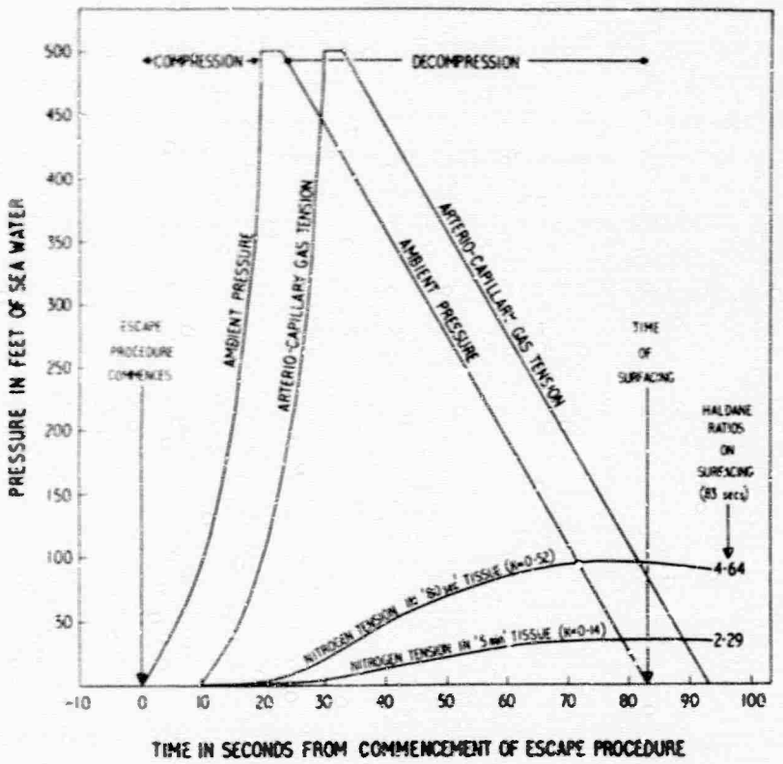

Figure 1 Illustrating the time pressure relationships in a submarine escape (Upshot V type) from 500 ft. The calculated nitrogen tension in a 'very fast' (k = 0.52) and 'fast' tissue (k = 0.14) are also shown during the escape cycle. (see text and Annex).

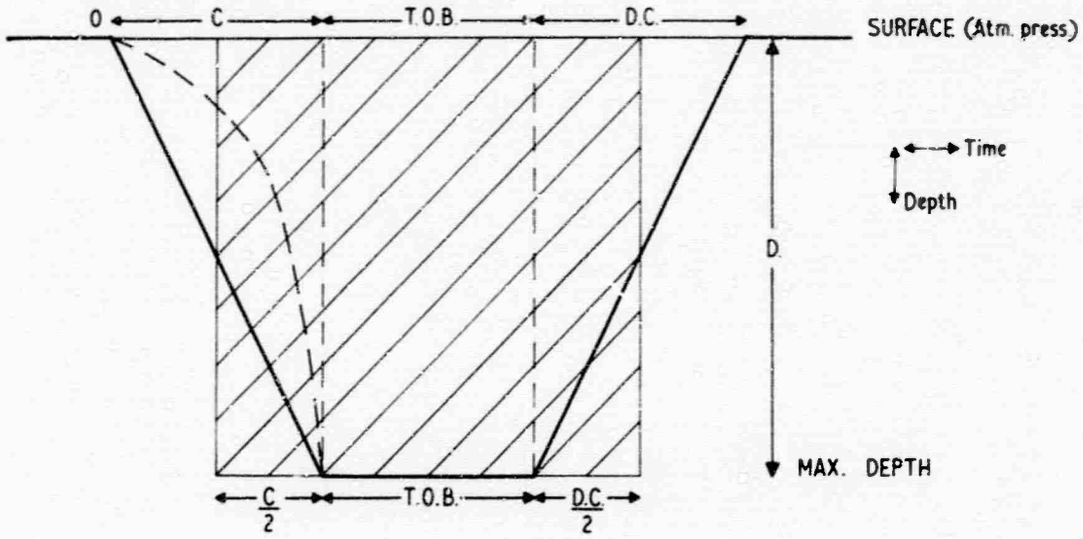

Figure 2 Diagramatic representation of time-pressure relationships during simulated or real submarine escape (heavy line). C = time of compression, T.O.B. = time on bottom, D.C. = time of decompression. The interrupted line shows the relationship with doubling of pressure in constant time as in Upshot IV and V. (see text).

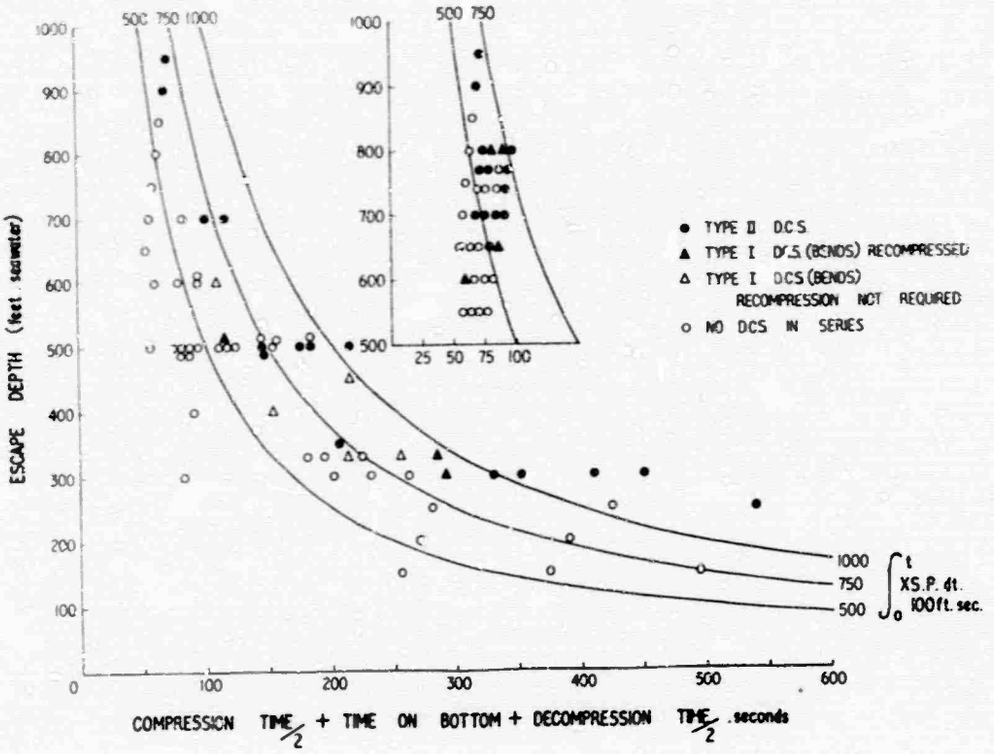

Figure 3 The escape depth is plotted against the 'equivalent time' at full depth (C/2 + T.O.B. + D.C./2). The isopleths represent the multiple of these values and equals $\int_0^t$ Excess P. dt (SPdt). All goat experiments from 1945 to 1970 are illustrated. Eaton's 1967(ii)studies are shown in a separate panel along with the goat studies from 650 to 950 ft. carried out before Upshot V. There is only one symbol for each group of goats with a particular escape profile. (see text and table).

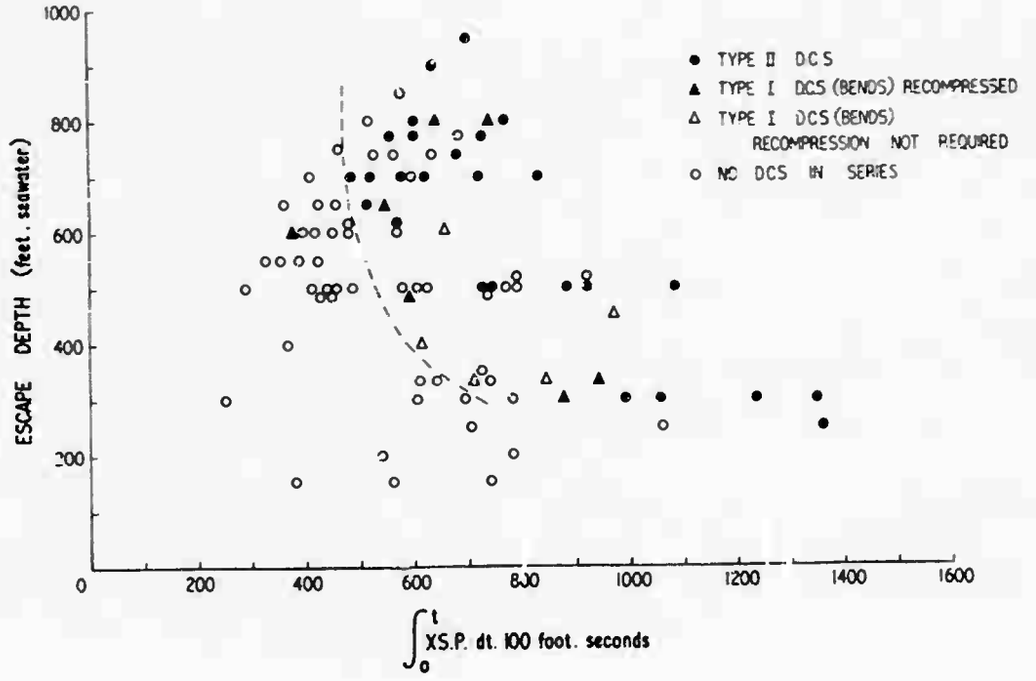

Figure 4 The escape depth is plotted against the calculated $\int_0^t$ Exeess pressure dt. All goat experiments from 1945 to 1970 are illustrated. There is only one symbol for each group of goats with a particular escape profile (see text and table).

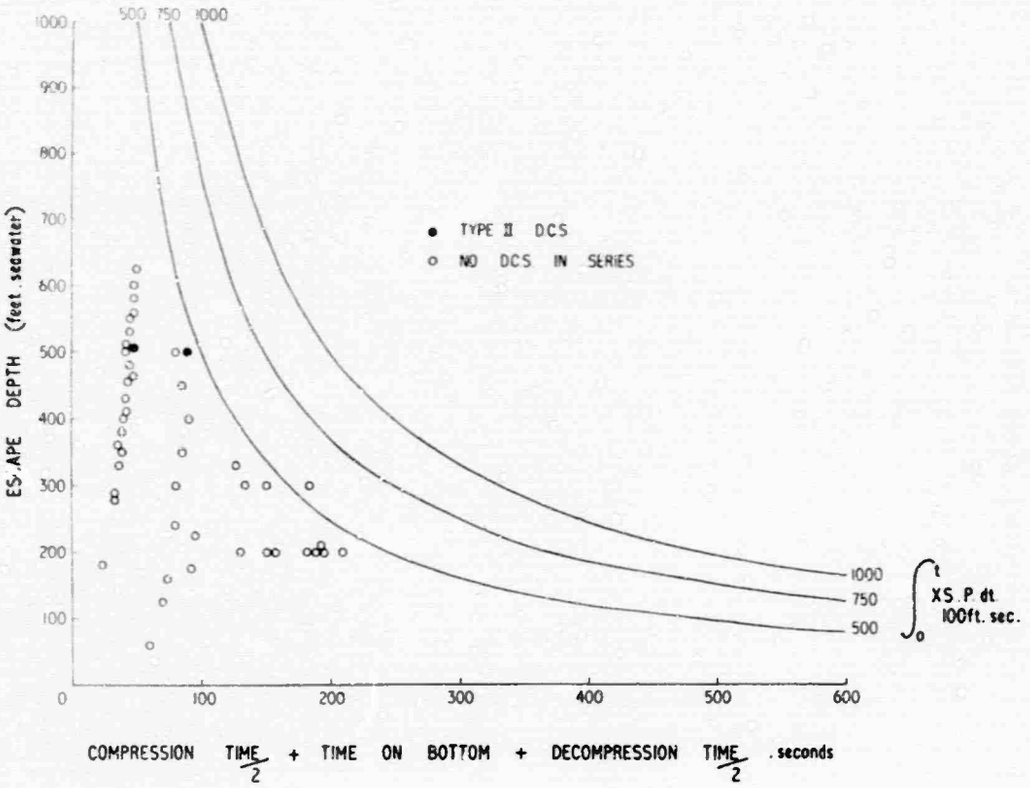

Figure 5 Human exposures only (see Table II). The escape depth is plotted against the 'equivalent time' at full depth (C/2 + T. O. B. + D. C. /2). The isopleths represent the multiple of these values and equal $\int_0^t$ Excess P. dt. (SPdt). The only two cases of post-escape decompression are discussed in the text. There is only one symbol for each group with a particular escape profile.

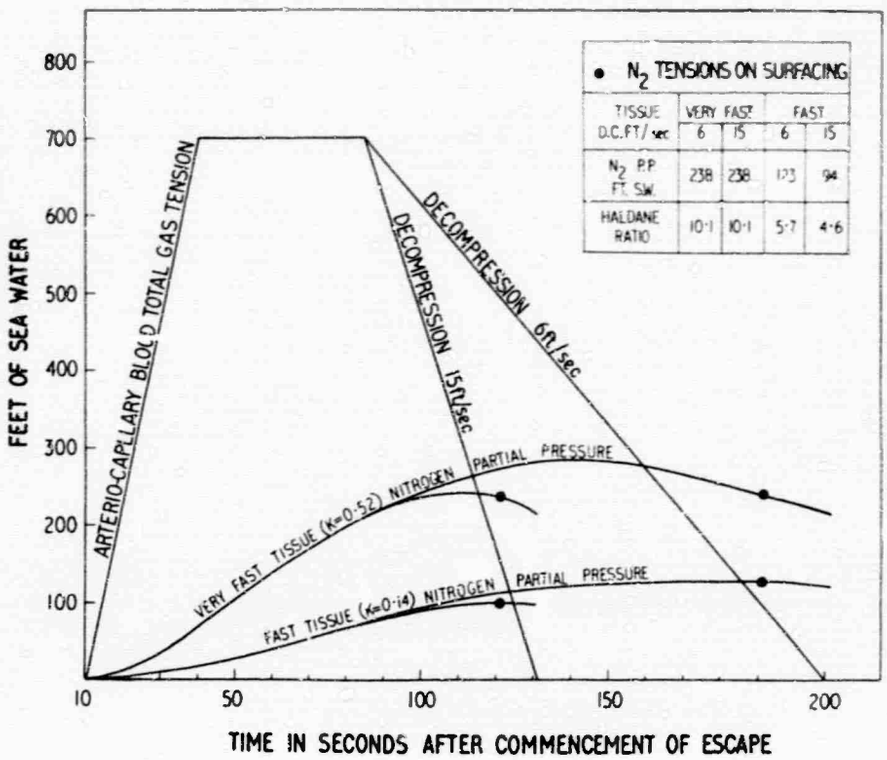

Figure 6 Illustrating the time pressure relationships in a 700 ft. escape (Eaton, 1967(i)) with widely differing rates of ascent (6 ft. per sec. and 15 ft. per sec.). The calculated nitrogen tensions in the 'very fast' (k = 0.52) and 'fast' (k = 0.14) tissues are shown in both escapes. There were three fatal cases of D.C.S. out of six in the 6 ft. per sec. ascent escapes and no cases of D.C.S. in eight 15 ft. per sec. ascent escapes.
Only the blood gas tensions are shown, the ambient pressure changes are about 10 seconds earlier. (see Figure 1).

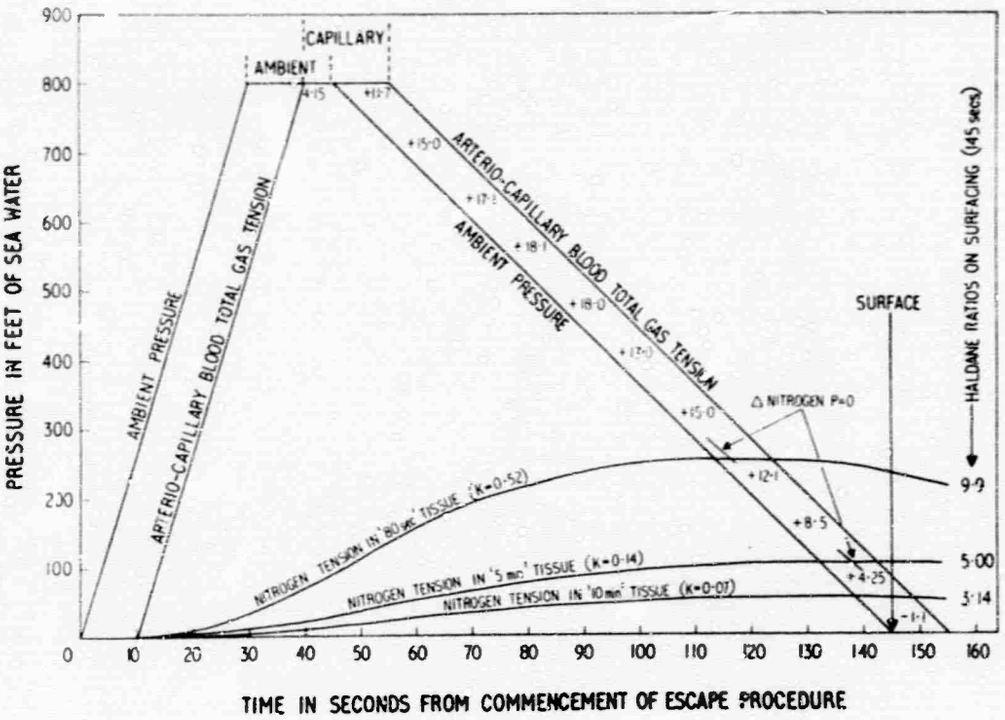

Figure 7 Illustrating the time pressure relationships in an 800 ft. escape (Eaton, 1967 (ii)). The calculated nitrogen tensions in '80 second', '5 minute' and '10 minute' tissues are shown during the escape cycle. Only one out of eight goats developed D. C. S. after this escape.
The figures along the arterio-capillary blood gas tension line show the excess of dissolved oxygen (in ml.) over oxygen consumed in one kilogramme of '80 second' tissue with an oxygen uptake of 33 ml. /Kg. /min. (see text).

TABLE I

DECOMPRESSION SICKNESS IN SIMULATED SUBMARINE ESCAPES BREATHING AIR (GOATS) 1945-1970.

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
DEPTH (Ft. Sea Water)	TIME ON BOTTOM (Sec.)	RATE OF COMP. (Ft./Sec.)	RATE OF DECOMP. (Ft./Sec.)	$\frac{C}{2}$: T.O.B. : $\frac{D.C.}{2}$ (Sec.)	EQUIVALENT TIME ON BOTTOM (TOTAL OF (5)) (Sec.)	TIME PRESS INDEX (SNIt × (6) × (3)) (100 Ft./Sec.)	NO. OF EXITS.	NO. WITH D.C.S.	DETAILS	DIAGNOSIS	RESULT
				DONALD, DAVIDSON and SHELFORD (1945)							
150	180	2	2	38 180 37	255	382	5	0			
	300	2	2	38 300 37	375	561	5	0			
	420	2	2	38 420 37	495	740	3	0			
200	180	2	2	45 180 45	270	540	5	0			
	300	2	2	45 300 45	390	780	6	0			
250	135-175	2	2	63 157 62	282	705	6	0			
	300	2	2	63 300 62	425	1062	5	0			
	470	2	2	63 420 62	545	1362	4	1	Case 1 Bend one leg. Therapeutic R.C. Flaccid paralysis hind legs after D.C. Killed.	B M	KILLED
300	180	2	2	75 180 75	350	880	5	3	Case 2 Chokes. Bends two legs. Immediately fatal.	Ch B	DIED
									Case 3 Chokes. ?Multiple bends. ?Paralysis R.C. Incurable. Killed.	Ch B	KILLED
									Case 4 Flaccid paralysis hind legs. Died during R.C.	M	DIED
	300	2	2	75 300 75	450	1350	5	2	Case 5 Bent one leg. Therapeutic R.C. Cured.	B	C.R.
									Case 6 Flaccid paralysis hind legs. Therapeutic R.C. Ineffective. Killed.	M	KILLED
						PRATT and TAYLOR (1946)					
330 (Reported 300)	30	2	2	83 30 82	195	683	6	0			
	60	2	2	83 60 82	225	742	10	0			
	90	2	2	83 90 82	255	841	14	2	Two cases (1 + 2) of mild bends not requiring R.C.	B B	C.R. C.R.
	120	2	2	83 120 82	285	940	14	3	Two cases (3 + 4) of mild bends not requiring R.C.	B B	C.R. C.R.
									Case 5 Bend requiring R.C. Cured.	B	C.R.
	60	2	1	83 60 41	184	610	12	0			
	90	2	1	83 90 41	214	706	13	2	Two cases (6 + 7) of very mild bends not requiring R.C.	B B	C.R. C.R.
						TAYLOR (1947)					
300	90	2	1	75 90 37	202	606	5	0			
	120	2	1	75 120 37	232	696	5	0			
	150	2	1	75 150 37	262	786	5	0			
	180	2	1	75 180 37	292	876	5	2	Case 1 Bend after 6 mins. R.C. Cured.	B	C.R.

27

300	180	2	2	75 180 75	330	990	5	3	Case 2 Chokes. Bends two legs. Immediately fatal.	Ch B	DIED
									Case 3 Chokes. ?Multiple bends. ?Paralysis R.C. Incurable. Killed.	Ch B	KILLED
									Case 1 Flaccid paralysis hind legs. Died during R.C.	M	DIED
	300	2	2	75 300 75	450	1350	5	2	Case 5 Bend one leg. Therapeutic R.C. Cured.	B	C.R.
									Case 6 Flaccid paralysis hind legs. Therapeutic R.C. ineffective. Killed.	M	KILLED
						PRATT and TAYLOR (1940)					
330 (Reported 300)	30	2	2	83 30 82	195	643	0	0			
	60	2	2	83 60 82	225	742	16	0			
	90	2	2	83 90 82	255	841	14	2	Two cases (1 + 2) of mild bends not requiring R.C.	B B	C.R. C.R.
	120	2	2	83 120 82	285	940	14	3	Two cases (3 + 4) of mild bends not requiring R.C.	B B	C.R. C.R
									Case 5 Bend requiring R.C. Cured.	B	C.R.
	60	2	4	83 60 41	184	610	12	0			
	90	2	4	83 90 41	214	706	12	2	Two cases (6 + 7) of very mild bends not requiring R.C.	B B	C.R. C.R.
						TAYLOR (1947)					
300	90	2	1	75 90 37	202	666	5	0			
	120	2	1	75 120 37	232	696	5	0			
	150	2	1	75 150 37	262	786	5	0			
	180	2	1	75 180 37	292	876	5	2	Case 1 Bend after 6 mins. R.C. Cured.	B	C.R.
									Case 2 Mild bend after 13 mins. R.C. Cured.	B	C.R.
	240	2	4	75 240 37	352	1056	7	6	Case 3 Bend. R.C. Cured.	B	C.R.
									Case 1 Flaccid paralysis. R.C. Failed. Killed.	M	KILLED
									Case 5 Mild bend. R.C. Cured.	B	C.R.
									Case 6 Mild bend. R.C. Cured.	B	C.R.
									Case 7 Very mild bend. Not R.C. Recovered.	B	C.R.
									Case 8 Very mild bend. Not R.C. Recovered.	B	C.R.
	300	2	4	75 300 37	412	1236	1	1	Case 2 Flaccid paralysis. R.C. Failed. Killed.	M	KILLED
						BARNARD and EATON (1965) SERIES I					
350	151-157	14	4.5	12.5 155 39	206.5	723	4	0			
400	84-93	13	4	16 88 50	154	616	1	1	Case 1 ?Mild bend in both forelegs. Not R.C. Recovered in 10 mins.	B	C.R.
450	150-155	16	4.5	14 152 50	216	972	1	1	Case 2 Rolling of head. Slight leg bend. Not R.C. Recovered.	?B	C.R.
500	152-156	20	5	13 154 50	317	1085	2	1	Case 3 Bends two legs. R.C. Air 160°. Collapsed during R.C. R.C. 90°. Paralysed all four legs. Killed.	M	KILLED

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
				BARNARD and EATON (1965) SERIES II							
300	32-35	12	1.5	13 31 37	84	252	4	0			
400	28-33	13.5	1.5	15 31 46	92	368	4	0			
500	8	10	5	26 8 54	88	440	1	0			
	18	12	5	21 18 51	90	450	1	0			
	20	12.5	5.5	20 20 15	85	425	1	0			
	28	15.5	5	16 28 53	97	485	1	0			
	45	11	5	22 15 53	120	600	1	0			
	55	11.5	5	17 55 53	125	625	3	0			
	71	10	5	25 71 52	148	740	1	1	Case 1 Paralysis commencing in fore-legs. R.C. 165' Air. Recovered after 30 mins. at 165'. Paralysis returned during D.C. Killed.	M	KILLED
	85	14.5	5	17 85 53	155	775	1	0			
	88	16	5	16 88 53	157	785	1	0			
	95	20	5	13 95 50	158	790	1	0			
	113	13.5	5	19 113 52	181	920	1	1	Case 2 Shivering hind legs. Recovered. Then rotation of head and bend one leg. R.C. 100' Air. Complete relief. Developed paralysis hind legs during D.C. after 2 hours. R.C. Killed.	M D	KILLED
	118	16	5	16 118 50	181	920	1	0			
				EATON (1967) 30 sec. compression assumed in all cases.							
500	30	17	6	15 30 42	87	435	6	0			
	30	17	20	15 30 12.5	57.5	287	6	0			
	60	17	6	15 60 42	117	585	6	0			
	60	17	20	15 60 12.5	87.5	437	6	0			
	90	17	6	15 90 42	147	735	8	3	Case 1 Bends two legs. R.C. 100' Air. Cured.	B	C.R.
									Case 2 Paralysed hind quarters R.C. 165' Air. Recurred on D.C. Kil[illegible].	M	KILLED
									Case 3 Bends two legs. R.C. 165' Air. Paralysed hind legs during D.C. left fore-leg weak. R.C. 100' Air. Recurred on D.C. Killed.	M	KILLED
	90	17	20	15 90 12.5	117.5	587	8	1	Case 4 Bend one leg. R.C. 100'. Cured.	B	C.R.
	120	17	20	15 120 12.5	147.5	737	3	0			
	150	17	20	15 150 12.5	177.5	887	1	1	Case 5 'Serious bends'. Collapse. No details. R.C. Air. Not cured. Assumed to be paralysed.	M	KILLED
600	30	20	6	15 30 50	95	570	12	0			
	30	20	15	15 30 20	65	390	12	0			
	45	20	6	15 45 50	110	660	8	1	Case 6 Bend one leg. Very distressed. No R.C. available. Recovered without R.C.	B	C.R.
	45	20	15	15 45 20	80	480	8	0			
	60	20	15	15 60 20	95	570	6	1	Case 7 Acute chokes 4 mins. after. Collapsed and died almost immediately.	Ch	DIED
700	30	23	6	15 30 58	103	721	12	1	Case 8 Paralysed. R.C. 165' Air. Failed. Killed.	M	KILLED
									Case 9 30 mins. after dive goat collapsed and died.	?C.N.S. ?Ch	DIED

	28	15.5	5	16 28 53	97	485	1	0			
	45	11	5	22 45 53	120	600	1	0			
	55	11.5	5	17 55 53	125	625	3	0			
	71	10	5	25 71 52	148	740	1	1	Case 1 Paralysis commencing in fore-legs. R.C. 165' Air. Recovered after 30 mins. at 165'. Paralysis returned during D.C. Killed.	M	KILLED
	85	14.5	5	17 85 53	155	775	1	0			
	88	16	5	16 88 52	157	785	1	0			
	95	20	5	13 95 50	158	790	1	0			
	113	13.5	5	19 113 52	181	920	1	1	Case 2 Shivering hind legs. Recovered. Then rotation of head and bend one leg. R.C. 100' Air. Complete relief. Developed paralysis hind legs during D.C. after 2 hours. R.C. Killed.	M D	KILLED
	118	16	5	16 118 50	184	920	1	0			
				EATON (1967) 30 sec. compression assumed in all cases.							
500	30	17	6	15 30 42	87	435	6	0			
	30	17	20	15 30 12.5	57.5	287	6	0			
	60	17	6	15 60 42	117	585	8	0			
	60	17	20	15 60 12.5	87.5	437	6	0			
	90	17	6	15 90 42	147	735	8	3	Case 1 Bends two legs. R.C. 100' Air. Cured.	B	C.R.
									Case 2 Paralysed hind quarters. R.C. 165' Air. Recurred on D.C. Killed.	M	KILLED
									Case 3 Bends two legs. R.C. 165' Air. Paralysed hind legs during D.C. Left fore-leg weak. R.C. 100' Air. Recurred on D.C. Killed.	M	KILLED
	90	17	20	15 90 12.5	117.5	587	8	1	Case 4 Bend one leg. R.C. 100'. Cured.	B	C.R.
	120	17	20	15 120 12.5	147.5	737	3	0			
	150	17	20	15 150 12.5	177.5	887	1	1	Case 5 'Serious bends'. Collapse. No details. R.C. Air. Not cured. Assumed to be paralysed.	M	KILLED
600	30	20	6	15 30 50	95	570	12	0			
	30	20	15	15 30 20	65	390	12	0			
	45	20	6	15 45 50	110	660	8	1	Case 6 Bend one leg. Very distressed. No R.C. available. Recovered without R.C.	B	C.R.
	45	20	15	15 45 20	80	480	8	0			
	60	20	15	15 60 20	95	570	6	1	Case 7 Acute chokes 4 mins. after. Collapsed and died almost immediately.	Ch	DIED
700	30	23	6	15 30 58	103	721	12	4	Case 8 Paralysed. R.C. 165' Air. Failed. Killed.	M	KILLED
									Case 9 30 mins. after dive goat collapsed and died.	?C.H.S. ?Ch	DIED
									Case 10 Bend one leg. Disturbed respiration. R.C. Air 100'. Cured.	B ?Ch	C.R.
									Case 11 Bend one leg. Paralysis. R.C. 100' Air. Failed. Killed.	B M	KILLED
	45	23	6	15 45 58	118	826	6	3	Case 12 Paralysis hind legs. R.C. 105' Air. Failed to alleviate. Killed.	M	KILLED
									Case 13 Paralysis and total collapse. R.C. 165' Air. Ineffective. Killed.	M	KILLED
									Case 14 Paralysis hind-quarters. R.C. Ineffective. Killed.	M	KILLED
	45	23	15	15 45 24	84	588	8	0			

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
						EATON (1957 11)					
550	10	18	8	15 10 34	59	324	30	0			
	10	18	6	15 10 46	71	390	29	0			
	15	18	8	15 15 31	64	352	29	0			
	15	18	6	15 15 46	76	118	29	0			
600	10	20	8	15 10 37.5	62.5	375	29	1	Case 1 Bends 3 legs. Dyspnoea. Collapse. R.C. 60' O_2. Immediate recovery. D.C. after 5 mins. Diagnosis dismissed.	B Ch M	C.R.
	10	20	6	15 10 50	75	450	29	0			
	15	20	8	15 15 37	67	405	29	0			
	15	20	6	15 15 50	80	480	29	0			
650	10	22	8	15 10 40	65	422	29	0			
	10	22	6	15 10 54	79	513	28	2	Case 2 Bends 2 legs. R.C. O_2 60'. Recovered.	B	C.R.
									Case 3 Rotation to left. General paralysis, R.C. Air 90-.60-96-82-55'-bleed to A.P. Weak and ill. Died.	M OTHER CNS	DIED
	15	22	8	15 15 40	70	455	26	0			
	15	22	6	15 15 54	84	546	26	1	Case 4 Bends 3 legs. R.C. O_2 60' for 3 hrs. 41 mins. Recovered.	B	C.R.
700	10	23	8	15 10 44	69	483	24	1	Case 5 Bends both fore-legs and one hind leg. Dyspnoeic. Collapsed. Paralysed. R.C. 60' O_2. Rapid recovery.	B M Ch	C.R.
	10	23	6	15 10 58	83	581	24	3	Case 6 Bends 2 legs. Improved. R.C. 60'O_2 16 mins. Bled 1 hr. ?Diagnosis	B	C.R.
									Case 7 Weak in hind legs. Distressed Short R.C. 60'O_2. Recovered. ?Diagnosis.	M	C.R.
									Case 8 Paralysed hind legs. Improved. Appeared normal without D.C. Some residual dragging hind legs next day.	M	Partial Recovery
	15	23	8	15 15 44	74	518	22	1	Case 9 Hind legs and one fore-leg paralysed. R.C. 60' O_2. Very rapid recovery. Normal after D.C. Dragging right hind leg next day.	M	P.R.
	15	23	6	15 15 58	88	616	21	1	Case 10 Bends and paralysis fore-legs. R.C. 30'O_2. On D.C. severe general paralysis. Killed.	B M	KILLED
740	10	25	8	15 10 46	71	525	19	0			
	10	25	6	15 10 61	86	636	17	0			
	15	25	8	15 15 46	76	562	19	0			
	15	25	6	15 15 61	91	673	17	2	Case 11 Bends two legs. Later paralysed. R.C. 60'O_2. Recovered.	B M	C.R.
									Case 12 Bends 2 legs. Collapsed with severe paralysis. R.C. O_2 60'. Recovered.	M	C.R.
770	10	26	8	15 10 18	73	562	19	1	Case 13 Weakness both hind legs. Collapse. R.C. 60'O_2. Walked normally after 3 mins. Recovered.	M	C.R.
	10	26	6	15 10 64	89	685	15	0			
	15	26	8	15 15 48	78	601	18	2	Case 14 Arched back. Severe paralysis. R.C. 60'O_2. Normal after few secs. Recovered.	M	C.R.
									Case 15 Severe paralysis hind legs. No R.C. available. Deteriorated. Killed.	M	KILLED
	15	26	6	15 15 64	94	724	15	1	Case 16 Bend one leg. Paralysis right hind	B M	C.R.

										dragging hind legs next day.		Recovery
	15	23	M	15 15 44	74	516	22	1	Case 9 Hind legs and one fore-leg paralysed. R.C. 60'O_2. Very rapid recovery. Normal after D.C. Dragging right hind leg next day.	M	C.R.
	15	23	G	15 15 58	88	616	21	1	Case 10 Bends and paralysis fore-legs. R.C. 60'O_2. On D.C. severe general paralysis. Killed.	D M	Killed
710	10	25	M	15 10 00	71	525	19	0			
	10	25	G	15 10 61	80	630	17	0			
	15	25	M	15 15 40	76	562	19	0			
	15	25	G	15 15 61	91	673	17	2	Case 11 Bends two legs. Later paralysed R.C. 60'O_2. Recovered.	R M	C.R
									Case 12 Bends 2 legs. Collapses with severe paralysis. R.C. O_2 60'. Recovered.	M	C.R.
770	10	26	M	15 10 58	73	562	19	1	Case 13 Weakness both hind legs. Collapse. R.C. 60'O_2. Walked normally after 5 mins. Recovered.	M	C.R.
	10	26	G	15 10 64	89	685	15	0			
	15	26	M	15 15 48	78	601	18	2	Case 15 Arched back. Severe paralysis. R.C. 60'O_2. Normal after few mins. Recovered.	M	C.R.
									Case 15 Severe paralysis hind legs. No R.C. available. Deteriorated. Killed.	M	Killed
	15	26	G	15 15 64	91	724	15	1	Case 16 Bend one leg. Paralysis right hind leg. R.C. 60'O_2. Recovered.	R M	C.R.
800	10	27	M	15 10 50	75	600	15	2	Case 17 Paralysis both hind legs and one fore-leg. R.C. 60'O_2. Normal in few minutes.	M	C.R.
									Case 18 Bends two legs. Chokes. Paralysis hind legs. R.C. 60'O_2. Recovered.	D Ch M	C.R.
	10	27	G	15 10 07	92	736	14	2	Case 19 Severe bend one leg. R.C. 60'O_2. Recovered.	R	C.R.
									Case 20 Bends 3 legs. ??Paralysis. R.C. 60'O_2. Recovered.	R	C.R.
	15	27	M	15 15 50	80	640	13	1	Case 21 Bends 2 legs. R.C. 60'O_2. Recovered.	R	C.R.
	15	27	G	15 15 07	97	776	12	3	Case 22 Chokes. Hind legs palsyed. R.C. 60'O_2. Recovered.	Ch M	C.R.
									Case 23 Bends 2 legs. R.C. 60'O_2. Recovered.	R	C.R.
									Case 24 Collapsed with paralysed hind legs. Res represaed 60'O_2. Dramatic recovery. Residual stiffness left hind leg.	M	P.R.

						SATOX (1 mg)			
650	5	26	8.5	12.5 5 30	55.5	341	12	0	
700	5	28	8.5	12.5 5 41	58.5	409	12	0	
750	5	30	8.5	12.5 5 41	61.5	401	12	0	
800	5	32	8.5	12.5 5 47	64.5	510	12	0	
850	5	31	8.5	12.5 5 50	67.5	576	12	0	
900	5	36	8.5	12.5 5 53	70.5	631	12	2	Nearly all animals appeared unwell. Two goats (1 and 2) with some degree of paralysis. Recovered without R.C.	M M	C.R. C.R.
950	5	38	8.5	12.5 5 56	73.5	698	12	3	All goats appeared unwell. Two goats (3 and 4) severe PNS. Not R.C. Killed.	M D	Killed Killed

TABLE 11

SIMULATED (CHAMBER) AND SEA TRIAL ESCAPES CARRIED OUT BY ROYAL NAVAL PERSONNEL 1945 - 1970

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
MAXIMUM DEPTH (FT. SEA WATER)	TIME ON BOTTOM (T.O.B.) (SEC.)	RATE OF COMPRESSION (C) (FT. PER SEC.)	RATE OF DECOMPRESSION (D.C.) (FT. PER SEC.)	$\frac{C}{2}$ T.O.B. $\frac{D.C.}{2}$ (SEC.)	'EQUIVALENT' TIME ON BOTTOM (TOTAL OF (5)) (SEC.)	TIME PRESS. INDEX (6) x (1) = UNIT (HUNDRED FT. SEC.)	NO. OF ESCAPES	COMMENT
			COWAN, WILSON, DICKSON and WRIGHT 1949 (CHAMBER)					
200	80	1.83	4.65	54.5 80 21.5	156	312	1	Compression and decompression approximately linear in time. All dives by Dr. Wright.
	130	1.73	4.75	57.5 130 21	208.5	417	1	
	105	1.6	4.07	62.5 105 24.5	192	384	1	
	110	1.64	4.25	61 110 23.5	194.5	389	1	
	112	2.1	4.53	47.5 112 22	181.5	262	1	
300	15	1.76	4.61	85.5 15 32.5	122	389	1	
				WRIGHT 1950 (CHAMBER)				
200	60	2.08	4.54	48 60 22	130	260	24	Compression and decompression approximately linear. Figures in brackets in column 8 are similar dives recorded but details are not available.
	60	2.17	2.22	40 60 45	151	302	8	
	60	2.74	1.08	36 60 93	189	378	2 (8)	
300	60	3.00	3.60	50 60 40	150	450	2 (28)	
	60	3.00	2.08	50 60 73	183	549	5 (12)	
330	30	3.00	4.00	55 30 41	126	416	2	
			H.M.S. TIRELESS AUGUST 1962 (SEA ESCAPES)					
125	17	N.L.	2	15 17 38	70	88	6	High pressure air equilibration. Latter half of compression much more rapid. Subjects decompressed in tower.
173	25	N.L.	2	16 25 51	92	161	2	
225	15	N.L.	2	16 15 61	95	214	4	
			H.M.S. TIPTOE SEPTEMBER 1962 (SEA ESCAPES) UPSHOT I					
60	40	N.L.	6	15 40 5	60	36	24	High pressure air equilibration. Compression non-linear. Pressure doubled in 10 - 12 secs. in last two series.
160	40	N.L.	5	16 40 17	72	117	12	
240	40	N.L.	5	16 40 23	79	190	12	

90	40	N.L.	6	15 40 5	60	36	36	
160	40	N.L.	5	16 40 17	73	117	19	High pressure air equilibration. Compression non-linear. Pressure doubled in 10 - 12 secs. in last two series.
240	40	N.L.	5	16 40 23	79	190	12	
				BARNARD and EATON 1965 (CHAMBER)				
300	40	15	5	10 40 30	80	240	10	
350	40	17.5	5	10 40 35	85	297	17	
400	40	20	5	10 40 40	90	360	10	Compression and decompression linear. °One type II D.C.S. in last series at 500 ft. Details reported in text.
450	30	22.5	5	10 30 45	85	382	19	
500	30	25	5	10 20 50	80	400	5	
	30	25	5	11.5 27 50	89.5	442	3°	
				H.M.S. ORPHEUS JULY 1965 (SEA ESCAPES) UPSHOT IV				
180	2	N.L.	8	11 3 11	24	44	10	
280	2	N.L.	8	14 3 17	33	99	9	
350	2	N.L.	8	15 3 23	39	136	10	Equilibration by flooding. Non-linear compression. Pressure doubled in 7 - 8 secs.
400	2	N.L.	8	13 3 25	40	160	10	
460	2	N.L.	8	14 3 29	46	207	9	
				BARNARD and EATON 1970(CHAMBER)				
500	3	25	9.5	10 3 39	42	310	6	
550	3	27.5	9.5	10 3 32	45	247	6	Compression linear.
600	3	30	9.5	10 3 35	48	288	8	
825	3	31	9.5	20 3 37	50	312	6	
				H.M.S. OSIRIS JULY 1970 (SEA ESCAPES) UPSHOT V				
280	3	x2/5 secs.	9.5	13 3 17	33	93	12	
330	3	x2/5 secs.	9.5	15 3 19	37	122	3	
350	3	x3/5 secs.	8.5	14 3 21	38	138	3	
380	3	x2/5 secs.	9.5	14 3 22	39	146	3	
405	3	x2/5 secs.	8.5	14 3 24	41	166	3	
430	3	x2/5 secs.	8.5	14 3 25	42	181	3	
455	3	x2/5 secs.	9.5	14 3 27	44	200	3	Equilibration by flooding. Pressure doubled approximately every 5 secs. °One type II D.C.S. in first series at 566 ft. For details see text.
480	3	x2/5 secs.	9.5	14 3 28	45	216	3	
505	3	x2/5 secs.	8.5	14 3 30	47	237	3°	
505	3	x2/5 secs.	8.5	12 3 30	45	227	3	
530	3	x2/5 secs.	8.5	11 3 31	45	239	3	
555	3	x3/5 secs.	8.5	11 3 33	47	261	3	
580	3	x2/5 secs.	9.5	11 3 34	49	279	3	

30 a

TABLE III

NITROGEN UPTAKE OF ONE KILOGRAMME OF TISSUE WITH 540 ml./min. PERFUSION OF BLOOD ('40 SECOND' HALF TIME TISSUE) DURING 500 FT. SUBMARINE ESCAPE

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	
TIME AFTER COMMENCEMENT (SEC.)	RANGE OF ART. CAP. BLOOD XS GAS TENSION (FT. S.W.)	MEAN OF (2) (FT. S.W.)	(3) IN (ATS.)	ART. CAP. BLOOD XS NITROGEN TENSION (ATS.)	INITIAL TISSUE XS NITROGEN TENSION (ATS.)	BLOOD TISSUE NITROGEN TENSION GRADIENT (ATS.)	EXCESS NITROGEN ENTERING TISSUE IN (ml./l. blood)	BLOOD FLOW PER Kg. OVER PERIOD (ml.)	TOTAL INPUT OF NITROGEN (ml.)	TISSUE FRACTION = 1000 / (1000 + BLOOD FLOW) (ml.)	INPUT OF NITROGEN INTO TISSUE (ml.)	SUMMATED INPUT OF NITROGEN INTO TISSUE (ml.)	TISSUE NITROGEN TENSION (ATS.)	TISSUE NITROGEN TENSION (FT. S.W.)	STAGE OF ESCAPE
10 - 25	0 - 230	77	2.33	1.85	0	1.85	22.0	135	3.00	0.88	2.70	2.7	0.22	7.3	COMPRESSION
25 - 30	230 - 500	320	9.68	7.67	0.22	7.45	90.8	45	4.08	0.90	3.90	6.6	0.54	17.8	
30 - 33	500	500	15.30	12.2	0.54	11.66	142	27	3.83	0.975	3.71	10.34	0.85	28.0	BOTTOM
33 - 43	500 - 417	458	13.90	11.0	0.85	10.15	124	90	11.10	0.915	10.32	20.56	1.68	55.4	
43 - 53	417 - 333	375	11.32	9.0	1.68	7.32	89	90	8.01	0.915	7.33	27.88	2.29	75.5	
53 - 63	333 - 250	291	8.83	7.0	2.29	4.71	57.3	90	5.10	0.915	4.72	32.60	2.67	88.3	DECOMPRESSION
63 - 73	250 - 167	209	6.33	5.0	2.67	2.33	28.4	90	2.56	0.915	2.34	34.94	2.86	91.0	
73 - 83	167 - 83	125	3.80	3.0	2.86	0.14	1.7	90	0.15	0.915	0.14	35.08	2.87	95.0	
*83 - 93	83 - 0	41.5	1.26	1.0	2.87	-1.87	*23.8	90		*0.085	-1.91	33.14	2.71	89.6	← SURFACE
					CALCULATION IGNORING INCREASING TISSUE NITROGEN TENSION (MINE)										
10 - 25	0 - 230	77	2.33	1.85			22.0	135	3.04	0.88	2.70	2.7	0.22	7.3	COMPRESSION
25 - 30	230 - 500	320	9.68	7.67	I	I	93.5	45	4.20	0.86	1.07	6.72	0.55	18.2	
30 - 33	500	500	15.30	12.2	G	G	148.3	27	4.00	0.975	3.90	10.62	0.87	28.8	BOTTOM
33 - 43	500 - 417	458	13.90	11.0	N	N	134.2	90	11.88	0.915	10.90	21.52	1.76	58.1	
43 - 53	417 - 333	375	11.32	9.0	O	O	109.8	90	9.88	0.915	9.05	30.55	2.50	82.7	
53 - 63	333 - 250	291	8.83	7.0	R	R	85.4	90	7.69	0.915	7.03	37.60	3.08	101.7	DECOMPRESSION
63 - 73	250 - 167	209	6.33	5.0	E	E	61.0	90	5.10	0.915	5.03	42.63	3.48	115	
73 - 83	167 - 83	125	3.80	3.0	D	D	36.6	90	3.28	0.915	3.02	45.65	3.71	123.7	← SURFACE
83 - 93	83 - 0	41.5	1.26	1.0			12.2	90	1.10	0.915	1.01	46.66	3.80	125.2	

*In this part of the ascent (83 - 93 secs.) tissue nitrogen exceeds blood nitrogen tension. 'Excess' nitrogen in the kilogramme of tissue is calculated (column 8) and partitioned to blood (note changed fraction)

TABLE IV

NITROGEN UPTAKE OF ONE KILOGRAMME OF TISSUE WITH 150 ml./min. PERFUSION OF BLOOD ('5 MINUTE TISSUE') DURING 500 FT. SUBMARINE ESCAPE

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	
TIME AFTER COMMENCEMENT (SEC.)	RANGE OF ART. CAP. BLOOD XS GAS TENSION (FT. S.W.)	MEAN OF (2) (FT. S.W.)	(3) IN (ATS.)	ART. CAP. BLOOD XS NITROGEN TENSION (ATS.)	INITIAL TISSUE XS NITROGEN TENSION (ATS.)	BLOOD TISSUE NITROGEN TENSION GRADIENT (ATS.)	EXCESS NITROGEN ENTERING TISSUE IN (ml./l. blood)	BLOOD FLOW PER Kg. OVER PERIOD (ml.)	TOTAL INPUT OF NITROGEN (ml.)	TISSUE FRACTION = 1000 / (1000 + BLOOD FLOW) (ml.)	INPUT OF NITROGEN INTO TISSUE (ml.)	SUMMATED INPUT OF NITROGEN INTO TISSUE (ml.)	TISSUE NITROGEN TENSION (ATS.)	TISSUE NITROGEN TENSION (FT. S.W.)	STAGE OF ESCAPE

30 - 33	500	500	15.30	12.2	G	G	148.8	27	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
33 - 43	500 - 417	458	13.90	11.0	N	N	131.2	90	11.88	0.915	10.60	21.92	1.76	58.1
43 - 53	417 - 333	375	11.32	9.0	O	O	109.8	90	9.88	0.915	9.03	30.55	2.50	82.7
53 - 63	333 - 250	291	8.83	7.0	R	R	85.4	90	7.69	0.915	7.05	37.60	3.00	101.7
63 - 73	250 - 167	209	6.33	5.0	E	E	61.0	90	5.49	0.915	5.02	42.63	3.48	115
73 - 83	167 - 83	125	3.80	3.0	D	D	36.6	90	3.29	0.915	3.02	45.65	3.74	123.7
83 - 93	83 - 0	41.5	1.26	1.0			12.2	90	1.10	0.915	1.01	46.66	3.80	125.2

← [illegible] SURFACE

*In this part of the ascent (83 - 93 secs.) tissue nitrogen exceeds blood nitrogen tension. 'Excess' nitrogen in the kilograms of tissue is calculated (column 8) and partitioned to blood (note changed fraction)

TABLE IV

NITROGEN UPTAKE OF ONE KILOGRAMME OF TISSUE WITH 150 ml./min. PERFUSION OF BLOOD ('5 MINUTE TISSUE') DURING 500 FT. MARINE ESCAPE

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	
TIME AFTER COMMENCEMENT (SEC.)	RANGE OF ART. CAP. BLOOD XS GAS TENSION (FT. S.W.)	MEAN OF (2) (FT. S.W.)	(3) IN (ATS.)	ART. CAP. BLOOD XS NITROGEN TENSION (ATS.)	INITIAL TISSUE XS NITROGEN TENSION (ATS.)	BLOOD TISSUE NITROGEN TENSION GRADIENT (ATS.)	EXCESS NITROGEN ENTERING TISSUE IN (ml./l. blood)	BLOOD FLOW PER Kg. OVER PERIOD (ml.)	TOTAL INPUT OF NITROGEN (ml.)	TISSUE /FRACTION 1000 / 1000 ÷ BLOOD FLOW (ml.)	INPUT OF NITROGEN INTO TISSUE (ml.)	SIMULATED INPUT OF NITROGEN INTO TISSUE (ml.)	TISSUE NITROGEN TENSION (ATS.)	TISSUE NITROGEN TENSION (FT. S.W.)	STAGE OF ESCAPE
10 - 25	0 - 230	77	2.33	1.85	0	1.85	22.0	37.5	0.85	0.955	0.82	0.82	0.07	2.3	COMPRESSION
25 - 30	230 - 500	320	9.68	7.37	0.07	7.60	93.0	12.5	1.16	0.99	1.14	1.96	0.16	5.3	
30 - 33	500	500	15.30	12.2	0.16	12.04	147	7.5	1.10	0.99	1.09	3.05	0.25	8.3	BOTTOM
33 - 43	500 - 417	458	13.90	11.0	0.25	10.75	131	25.0	3.27	0.975	3.18	6.23	0.51	16.8	
43 - 53	417 - 333	375	11.32	9.0	0.51	8.49	103	25.0	2.58	0.975	2.53	8.76	0.72	22.7	
53 - 63	333 - 250	291	8.83	7.0	0.72	6.28	76.6	25.0	1.91	0.975	1.86	10.62	0.87	28.7	
63 - 73	250 - 167	209	6.33	5.0	0.87	4.13	50.5	25.0	1.26	0.975	1.23	11.85	0.97	32	DECOMPRESSION
73 - 83	167 - 83	125	3.80	3.0	0.97	2.03	24.8	25.0	0.62	0.975	.60	12.45	1.02	33.6	
83 - 93	83 - 0	41.5	1.26	1.0	1.02	0	0	25.0	0	0.975	0	12.45	1.02	33.6	← SURFACE
					CALCULATION IGNORING INCREASING TISSUE NITROGEN TENSION (SIMI)										
10 - 25	0 - 230	77	2.33	1.85			22.0	37.5	0.85	0.955	0.82	0.82	0.07	2.3	COMPRESSION
25 - 30	230 - 500	320	9.68	7.37	I	I	93.7	12.5	1.17	0.99	1.16	1.98	0.16	5.1	
30 - 33	500	500	15.30	12.2	G	O	149	7.5	1.12	0.99	1.11	3.09	0.25	8.3	BOTTOM
33 - 43	500 - 417	458	13.90	11.0	N	N	131	25.0	3.35	0.975	5.27	8.36	0.52	17.1	
43 - 53	417 - 333	375	11.32	9.0	O	O	100.8	25.0	2.75	0.975	2.68	9.04	0.74	24.4	
53 - 63	333 - 250	291	8.83	7.0	R	R	85.5	25.0	2.14	0.975	2.09	11.13	0.91	29.8	DECOMPRESSION
63 - 73	250 - 167	209	6.33	5.0	E	E	61	25.0	1.52	0.975	1.48	12.61	1.03	34.1	
73 - 83	167 - 83	125	3.80	3.0	D	D	36.6	25.0	0.91	0.975	.87	13.5	1.10	36.4	
83 - 93	83 - 0	41.5	1.26	1.0			24.4	25.0	0.61	0.975	.59	14.1	1.16	38.4	← SURFACE

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